



CHEMISTRY, SAMPLING AND ANALYSIS OF MERCURY

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Ministry
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CHEMISTRY, SAMPLING AND ANALYSIS OF MERCURY

By:

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Presented at the
Thunder Bay Mercury Seminar
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Chemistry, Sampling and Analysis of Mercury

A: HISTORY

To introduce the topic of mercury and its chemistry, I would like to follow two main aspects of its history: The purely anecdotal history, and its history in Ontario as a pollutant.

History is full of references to mercury and its uses, and such references date back to the beginning of recorded history itself. These ancient references to mercury are generally typified by a good deal of confusion as to exactly what the metal and its compounds were good for. For example, the ancient Chinese believed that it would prolong life, while the ancient Hindus valued it as an aphrodisiac. Although several Chinese emperors died showing classical symptoms of mercury poisoning (1), little is recorded of the ultimate fate of early Hindus testing their particular theory.

The Romans around the time of Hannibal were using the principal ore of mercury, cinnabar, as a rouge and general beauty product. The use of vermillion, a reduced form of cinnabar, continues today as a high grade paint pigment. The Romans also mined mercury to use in recovering gold in various processes. They were not completely unaware of its toxicity, even then, as Hippocrates, Galen, and other physicians had long warned of the poisonous nature of mercury. The Romans, being practical people, acknowledged this fact by sending slaves who were condemned to death to work in the mercury mines. (2)

The next big impact mercury had on the public was around 1530 A.D., when Paracelsus, the Swiss alchemist and physician developed a "cure" for syphilis which involved a mixture of mercury, sulfuric acid, and alcohol. Presumably the mixture was applied externally, and the afflicted organ would either heal or fall off. In any event, it is interesting to note that mercury compounds were used in the treatment and cure of syphilis as recently as the 1930's, in spite of their apparent ineffectiveness and undesirable side effects. Many of the symptoms of syphilis as noted in seventeenth and eighteenth century medical books correspond closely with descriptions of mercury poisoning. It has been suggested that perhaps the cure became the disease. (3)

Mercury and its salts were used in treatment of many other diseases, yielding symptoms essentially the same as some forms of syphilis, and of Minamata disease. The Toronto Star, in a special report on November 6, 1971, disclosed some interesting data concerning Robert Burns. This Scottish national, long believed to have died from excessive drink and wild wenching, may have died from mercury poisoning.

He was known to be on mercury medication, and his hair was analyzed last year, showing abnormally high levels of the metal. (4)

Getting back to Paracelsus and the Middle Ages, mercury now became the spotlight for the alchemists. The alchemists are the venerable ancestors of the modern chemists and are far from extinct; in fact, most of us probably have a soft spot for them and secretly admire them. Their fascination with mercury was the result of Paracelsus' theory of metals (5) which was quite logical: all metals have an end product, which is coagulation. Therefore, gold is just mercury which has become coagulated. And if someone could find a way to coagulate mercury, he would naturally convert it to gold, and while he would not win the Nobel Award, he would become fabulously wealthy. For 200 years, our alchemist ancestors worked on this transmutation theory.

In 1557 the Patio process of gold and silver refining was invented. This involves amalgamating these metals with mercury, then heating the amalgam to drive off mercury, leaving the precious metals behind. Spain at this time controlled what is today Mexico, Peru, Bolivia, etc., as well as most of the world's seas, and they owned the largest mercury mine in the world, at Almaden. Tremendous amounts of mercury were shipped to the America's for the next 300 years, and many shiploads were likely sunk during battles and storms. In 1810, the British warship Triumph salvaged a cargo of mercury from a disabled Spanish vessel. The mercury was transferred from sealed drums on the Spanish ship to wooden barrels on board the Triumph. By the time the Triumph got to port, at least three sailors had died of acute mercury intoxication, and 200 others were severely poisoned. (6)

At about this same time, an even greater mass mercury poisoning occurred in Austria. (6) A fire in a mercury mine created vapours that left 900 locals with mercurial tremors. Swine, dogs and cows in the area were similarly affected.

Occupational mercury poisoning had been observed in miners for centuries (7), but the wholesale poisoning of a city or a warship introduced a new dimension to the problem. Other instances of occupational intoxication by mercury were observed in the felt hat industry, where symptoms gave rise to the term "Mad Hatter" (8), and in criminology where policemen who used mercury powder for developing fingerprints often developed tremors of the hands and feet (9), and also in dentists via mercury vapor inhalation from dental amalgams.

However, these cases are dwarfed by subsequent mass poisonings within the past few decades. These can be divided into two types: where the epidemic is caused by direct use or misuse of alkylmercury-containing fungicides, and where mercury or methylmercury is released into the environment as a by-product and are subsequently concentrated into the human food chain. I will deal with the former type first. In 1961, about 100 people in Pakistan (10) were afflicted with organomercurial poisoning. In northern Iraq in 1956 more than 100 cases of mercury poisoning were reported, with fourteen deaths. Four years later, near Baghdad, Iraq, many farmers were poisoned, and 221 patients were admitted to just one hospital in Baghdad. There were many deaths, and in 22 bodies examined, in the Medical-Legal Institute in Baghdad, large amounts of mercury were found in the internal organs (average liver level = 68.5 ± 18.5 ppm) (11). In Guatemala (12) 45 cases of organomercurial poisoning resulted in twenty deaths in 1965. And in 1972, there were 6530 (officially) cases of mercury poisoning in the tragic Iraq incident, while the private estimates of experts suggest that as many as 6000 may have died, and perhaps 100,000 may have been injured (13).

These cases were all apparently the result of people eating organomercurial-dressed wheat. Some of the patients washed the wheat to rid it of the poison. It appears that in most cases, the people had been warned against eating the grain, but when they observed that nothing happened to fowl that ate the grain, they also consumed it. Some of them appeared healthy for days, thus providing an unfortunate example for the others, who began to eat freely of the contaminated seed. (14)

In September 1969, a granary in New Mexico gave away floor sweepings, and a custodial worker collected some 3,000 pounds to feed his hogs. About three weeks after he began feeding them this diet, he noticed that a number of the hogs appeared sick. He subsequently butchered the largest one that appeared well, and began feeding the meat to his family. About three months later, three of his children developed impaired vision and eventually became comatose. Urine analysis (15) revealed high mercury contents and hair samples from two of the children contained 1,397 and 2,436 ppm mercury (these being the highest levels ever measured in a human). Six months later, two of the children were still blind (one in a coma) and the third appeared to be recovering, although with some impairment of vision. Checks revealed that the seed at the granary had been treated with Panogen, a methylmercury formulation. (11)

These mass poisonings are examples of the inadvertant or accidental direct poisoning by organomercurials. The second type of poisoning, that caused by accidental leakage of mercury or methylmercury to the environment and subsequent uptake into the human diet, occurred in Japan.

The first incident occurred in Minamata, an industrial city of 50,000, located on the coast of the most southerly of the main Japanese islands. The main industry of the community is a fertilizer factory, which in 1949 began to produce vinyl chloride and acetaldehyde by means of catalytically converting acetylene. The catalyst employed was mercury. The factory dumped the plant effluent into Minamata Bay, where the methylmercury in the effluent began to contaminate the fish and shellfish. Minamata Bay provided seafood for many families near the shoreline, and a large number of people gradually became poisoned. It was noticed that cats became seriously ill, screamed incessantly, and often killed themselves by jumping into the sea. By the end of 1953 a mysterious disease that affected the central nervous system began to strike the Minamata Bay population. This grew to epidemic proportions by 1956. Initially the epidemic was believed to be caused by an infectious bacteria or virus transmitted from cats to humans. This peculiar syndrome was called Minamata Disease. A university group began to study the disease. (16)

When these investigators fed the cats fish that were caught in Minamata Bay, a cause and effect relationship was quickly established between the fish and the disease. Consequently, all fishing in Minamata Bay was banned in early 1957. On the basis of their results, the investigating team from Kumamoto University theorized that the causative agent was a heavy metal or a substance similar to a metal. During 1957 and 1958 many metals such as manganese, selenium, thallium, copper, zinc, lead and arsenic were investigated and ruled out. By the end of 1958 Professor Takeuchi had characterized the major pathological features of the disease, and had found them to be identical with the symptoms of organomercurial poisoning that were reported in the scientific literature in 1940. On July 14, 1959, two and a half years after the investigation began, the causative agent was identified as an organomercurial compound. Further investigation established that methylmercuric sulfide and methylmercuric chloride were present in the fish and shellfish of Minamata Bay. These compounds produced identical symptoms in experimental animals. (17, 18)

The process of determining the origin of these toxic materials in Minamata Bay proved equally difficult.

The most evident source of methylmercury compounds was determined to be the effluent from the fertilizer plant. However, the owners of this plant not only excluded scientists from their factories, but they also disseminated incorrect information about the source and conversion of the mercury catalyst that they were using. Ironically, the owners of the plant aided the research when they changed the factory's effluent drainage site. This change gave the Kumamoto University research group the opportunity to analyze and confirm that the effluent was responsible for the methylmercury compounds that were present in the fish and shellfish of Minamata Bay.

Finally, by 1964, eleven years after the first case of Minamata Disease was diagnosed, all of the pieces of the puzzle were fitted together. The researchers concluded that during the catalytic conversion of acetylene into vinylchloride, some of the mercuric chloride catalyst was unknowingly converted into a methylmercuric compound. This compound was carried into Minamata Bay in the plant's wastewater effluent. Subsequently, the methylmercury compound was biologically concentrated by the fish and shellfish of the bay and then transferred to the people of the area when they ate the fish. From the inception of the Minamata disease in 1953, until the present, a total of 1200 cases with 100 resulting deaths have been reported.

No sooner had the complete story of the Minamata disease been pieced together than the same problem was recognized at Niigata, Japan. As at Minamata, nearly all the victims lived near a water source, the Agano River; and they were heavy fish eaters. Based on the experience at Minamata Bay, the investigators quickly found a high concentration of organomercuric compounds in the fish and shellfish of the Agano River. In this case the responsible agency was a chemical plant that disposed of its wastewater effluent which contained organomercurials into the Agano River. As at Minamata the chemical plant executives made contradictory statements while the prefectural government not only claimed to be unaware of the problem, but they withheld information that was needed for the investigation. In Niigata a total of 47 cases were reported with six deaths. Therefore, the Minamata disease accounted for 1200 cases, and at least 106 deaths in the two incidents.

These horror stories are all quite true, and most of them occurred within the past decade or two. The truly horrible part of the whole problem is that the levels of mercury determined to be present in the fish that these people were eating were not significantly higher than for many fish caught in Canadian waters. For example, the data from the Niigata disaster indicate that consumption of 1.5 mg of mercury as methyl mercury per day (which is the

equivalent of 250 gm of fish 5-6 ppm (in mercury) would probably be lethal. (19) Consumption of similar amounts of fish from some Canadian waters would result in ingestion of similar amounts of methylmercury.

In Ontario, however, the history of mercury is not nearly as horrible. In fact, there have been no deaths attributable to mercury poisoning in this province, and there have not even been any cases of hospitalization as a result of mercury ingestion from fish or other foods.

However, this does not mean that Ontario does not have a mercury problem. There are two areas of the province where fish have been found to contain levels of mercury in the same range as found in the Japanese fish at Minamata. The question of just what level of mercury was responsible in the Minamata situation is not easily answered. First, there are differences in levels between species and between specimens of any given species. Then, there were analytical problems. For example, the Japanese papers of 1960's all stated that the amount of mercury present in fish or methyl mercury was between 10 and 60%. This is most likely due to shortcomings in the recovery of methyl mercury in the early Gas chromatograph methods, and could mean that the levels of methyl mercury responsible for the Japanese tragedies were much higher than claimed; that is, the fish contained 50-80 ppm minimum rather than 5-20 ppm. Thirdly, there was great confusion for several years in interpreting the Japanese problem because most of their data had been reported as "dry" weight. Thus, when levels of 50 and 80 ppm of mercury were reported in shellfish and finfish at Minamata, these levels were taken (in Sweden, mainly) to mean 50-80 ppm of mercury in fish as they are, in the water. Fish generally contain 75-80% H₂O, so the "dry" weight data must be divided by a factor four or five to render them meaningful in terms of live fish. Today the level of mercury in the fish of Minamata Bay in the 1950's is accepted as 10-15 ppm, subject to the reservations associated with the analytical problems mentioned earlier.

And it is this point that makes Ontario a province that is fortunate: there are some cases where fish have had mercury levels greater than 15 ppm in Ontario. That there have been no deaths because of mercury is probably more a result of Canadian eating habits rather than a cleaner environment. These areas and levels will be discussed later on.

In the summer of 1969, the Ontario Water Resources Commission advised Dow Chemical of Canada that mercury had been found in the sediments of the St. Clair River, below the outfall of their mercury cell chloralkali plant. Dow began appropriate plant modifications, which were scheduled for completion in May 1970. In the fall of 1969, hunting of some game birds in Alberta was banned because of mercury contamination, and in late November 1969, it was announced that fish taken from the South Saskatchewan River contained up to 10 ppm of mercury. This set the stage for one of the largest field and analytical programs ever undertaken for environmental purposes in Canada.

B: TOXICOLOGY OF MERCURY: 1. Introduction

As evidenced in the historical review of mercury, its threat to animal and human health has been well known since early times. However, illness and death caused by mercury or by inorganic mercury salts have been known since early times. However, organomercurials have only been known since 1914, hence their history as a toxicant is much more recent. There are three main classes of organomercurials, based upon the nature of the organic radical in the compound. Organomercurials are usually represented by the simplified formula $R-Hg-X$, where R is the organic radical and X represents an organic or inorganic substance possessing a dissociable hydrogen atom. X could be an inorganic acid radical (chloride, bromide, cyanide, etc.), an organic acid radical (acetate, benzoate, etc.), a phenolate, and so on. The organic radical R has a pronounced effect on an organometallic's toxicity.

There are three main groups:

- i Alkyl Mercury Compounds (methyl mercury, ethyl mercury)
- ii Alkoxyalkyl Mercury Compounds (methoxyethyl mercury, ethoxyethyl mercury)
- iii Aryl Mercury Compounds (phenyl mercury, p-tolyl mercury)

Since 1914 there have been over 60,000 cases of organomercurial poisoning and about 600 deaths (6,000 if the London Times is correct). (13)

2. Mercury in Fish: Epidemiology of Methyl Mercury Poisoning

There have been no documented cases of poisoning in human beings caused by fish consumption, except for the epidemics in Japan. It should be pointed out that in the Japanese epidemics, methylmercury itself was released from industry. The problem in various other countries has arisen out of concern regarding the possible effects of methylmercury in fish, but while high levels have been reported in fish taken in Sweden, Italy, U.S.A., and Canada, symptoms of methylmercury poisoning have only appeared in Japan.

In Minamata Bay and Niigata, Japan, the first indications that methylmercury in fish could constitute a public health threat arose in the 1950's and 60's. In what is now called "Minamata Disease", 112 people living in villages around Minamata were poisoned during the years 1953 to 1960. Twenty-two infants were poisoned pre-natally, and forty-six people died. At Niigata, 47 cases were documented and six deaths were recorded.

The symptoms of those afflicted have been carefully documented (16, 17, 18) and in approximate order of mild to severe intoxication they include:

- two discrete stages of motor impairment
- excessive fatigue, headache, general feeling of weakness, slight amnesia, inability to concentrate
- numbness and tingling of mouth, tongue, lips, hands, fingers, feet and toes (paresthesia)
- ataxia
- speech impairment
- loss of coordination, unsteadiness of gait
- reflex changes
- concentric narrowing of visual field ultimately leading to "gun-barrel" vision
- hearing impairment

- severe emotional disturbances (unpredictable violence, fits of extreme depression) ("erethism")
- paralysis
- death

Limits on the consumption of fish or on the level of mercury allowable in fish for human consumption, have been set by various means.

i) Berlin (20) used the following calculation:

Assuming a half-life of seventy days for methylmercury in humans (21), a daily intake of 1 mg mercury as methylmercury, "the body content at steady state would be about 100 mg Hg. If 15% of this is in the brain (21), i.e., 15 mg, and the brainweight is 1500 gm, the concentration in the brain should be 10 mg Hg/kg or 1000 ug/Hg gm." (22) This level is known to cause irreversible injury or death.

Conclusion: a daily intake of 1 mg of mercury as methylmercury will lead to toxic concentrations in the brain.

The people in Minamata consumed about 200 grams of fish per day, and the fish contained an average of 10 ppm mercury; they were consuming 2 mg mercury a day.

Assuming 1 mg per day as a toxic dose, Berlin et al (20) used a safety factor of 10, making the upper limit of the ADI 0.1 mg per day. A diet of 100 gm (2/5 lb.) per day of St. Clair fish at 1 ppm would approach this level. Since many fish in the St. Clair and other systems are higher than 1 ppm, there exists a potentially grave health problem in these areas. It is worth noting that the 0.1 mg limit does not necessarily protect pregnant women or sensitive individuals.

ii) Dr. Stopps, in his report for the Ontario Department of Health, arrived at a level of 0.5 ppm as a "maximum permissible level" (23) of mercury in fish. This was based on the knowledge that the Japanese at Minamata Bay showed symptomatic effects of mercury poisoning with a daily intake of 2 mg (200 g of 10 ppm

fish). A safety factor of 10 is applied to this number to provide for variation in toxic response by members of a population. The 0.2 mg resulting from this is further factored by 4 to allow for use of a "severe effect" level rather than the usual "no effect" level. Thus 0.05 mg, or 50 ug, is the maximum permissible intake of mercury as methylmercury. If 100 grams of fish per day is used as the dietary average of high fish-consuming Canadians, the fish could contain up to $50 \text{ ug}/100 \text{ gm} = 0.5$ ppm mercury without exceeding this limit.

- iii) In a study of persons in Sweden (24) with a high intake of fish, one subject was found who consumed about 150 mg of fish per day. The fish had an average of 6.7 ppm mercury, so he was consuming about 1.0 mg mercury per day. A thorough clinical investigation revealed no signs or symptoms of illness, except his blood cells contained 1.2 ppm mercury (about half of that of persons afflicted with mercury poisoning). Thus, 1.0 mg mercury represents the highest intake ever calculated for an asymptomatic person, and 1.0 mg could be considered a "no effect" dose (based on one individual). The usual safety factor of 10 yields an ADI of 0.1 mg. This is double the level derived in Stopps' report. (23)
- iv) The metabolism and retention of (^{203}Hg) labelled methylmercury nitrate was studied in three adult males. (21) The half-life of mercury was found to be 70-74 days, or a daily excretion of 1% of the body burden. The amount of mercury in the brain which causes toxicity is about 12-15 mg. If this amount (12 mg) is assumed to be 10-20% of the total body burden, the latter should be 60-120 mg. With a daily excretion rate of 1%, the dose required to maintain equilibrium would be equal to 0.6-1.2 mg mercury as methylmercury per day. Applying a safety factor of 10 and assuming 0.6-1.2 mg mercury to be the minimum toxic dose yields an ADI of 0.06-0.12 mg mercury. This figure agrees with both Stopps' and the Swedish data.
- v) Lofroth in Sweden reviewed the subject of mercury (25) toxicity and recommends a maximum acceptable intake level of 0.03 mg per day. For a 154 pound (70 Kg) man, this works out to:
0.03 mg/70 Kg per day,
= 0.0004 mg per Kg per day,
= 0.4 ug per Kg per day.

Thus, a 154 pound male eating fish (200 gm per serving) containing 1 ppm Hg would consume 200 ug ($200 \text{ gm} \times 1 \text{ ug/gm} = 200 \text{ ug}$) mercury per meal.

His MAI is 30 ug. Accordingly, he should eat fish contaminated at this level no more often than once a week. Lofroth recommends a MAC in fish of 0.2 ppm in order to protect the public.

- 6) Clarkson, et al (26), established the Threshold Body Burden to be between 25-40 mg. The Swedish experimental group established it at 30 mg. Using this value, and assuming that 15% of the mercury would be located in the brain (21) there would be 4.5 mg of mercury, or approximately $\frac{1}{2}$ the amount known to cause irreversible injury or death, in the brains of people at the "threshold" level of mercury intoxication.
- 7) Summary of AID's, MAC's, and MAI's:

WORKER	ADI	FISH MAC	CONCLUSIONS
Berlin (20)	0.1 mg	-	1 mg mercury per day will cause mercury poisoning
Stopps (23)	0.05 mg	0.5 ppm	-
Birke (24)	0.1 mg	-	Found no effect with 1 mg Hg/day
Ekman (21)	0.06 - 0.12 mg	-	Half life of methylmercury in man: 70-74 days
Lofroth (25)	0.03 mg	0.2 ppm	-

- 8) 'Threshold body burden' refers to the onset of rigors and symptoms over and above the background level of such probabilities (5-10%). Clarkson's value was 25-40 mg of mercury. This is in excellent agreement with the Swedish figure of 30 mg from Japanese data.
- 9) In Clarkson's Iraq paper, there are certain symptoms associated with given mercury levels in patient's blood:

Hg in Blood (ug/ml)	% of Cases with Symptoms				
	Paraesthesia	Ataxia	Visual Changes	Hearing Defects	Death
0-100	9.5	5	0	0	0
101-500	5	0	0	0	0
501-1000	42	11	21	5	0
1001-2000	60	47	53	0	0
2001-3000	79	60	56	12.5	0
3001-4000	82	100	58	36	17
4001-5000	100	100	83	66	28

Thus it appears that some threshold's are available: at 500 to 1000, paresthesia, ataxia, and visual changes take place. Also, at 3000 and up, all symptoms including death, are noticeable.

C: MERCURY PROGRAMS IN ONTARIO

The Province of Ontario has, since the problem first came to public attention in 1969, undertaken a massive mercury program. The Laboratory at O.W.R.C. and the MOE has been involved in many parts of this program.

Since 1970, we have analyzed over 35,000 fish, 12,000 sediments, and 15,000 waters as part of the following programs:

- I - Delineation of Extent of the Mercury Problem in Ontario.
See Tables 1, 2 and Figures 1 - 3.
- II - Summary of Mercury Concentrations in Fish and Sediments from the St. Clair System 1970-1973.

i - Introduction

During the period 1949-1970, the major contributor to mercury pollution in the St. Clair River was the Dow chlor-alkali complex at Sarnia. It is estimated that losses of mercury averaged 30 pounds per day since 1949 and reached a maximum of 75 pounds (average) per day in 1969. On March 26, 1970 the O.W.R.C. issued a Ministerial Order that Dow cease discharging mercury to the St. Clair River and since that time the losses of mercury have dwindled to less than one pound per day. At the present time Dow is no longer using the mercury cell at its Sarnia operations, having converted to the diaphragm cell within the past two years.

Analysis of sediments from the St. Clair River and Lake St. Clair (see map 4, figure 4) revealed extremely high levels of mercury (20-1200 ppm in the river, 8-12 ppm in the lake) and analysis of fish indicated that a serious problem existed in the St. Clair System. Accordingly, because fish levels for practically all species of commercial or recreational importance were well above the W.H.O. (World Health Organization) limit of 0.5 ppm mercury, commercial fishing was curtailed in April, 1970.

HUDSON BAY

Figure 1: Mercury
Concentration in Ontario
Northern Pike

MANITOBA

ONTARIO

JAMES
BAY

QUEBEC

Kenora
3.3 8.9 3.7
4.1 6.0 0.8

0.4

.3

0.3

Lake Timiskaming

1.1 LAKE
SUPERIOR

0.4

0.3

0.2

Lake Nipissing

0.2

0.4

0.8

0.9

0.6

0.5

0.7

0.7

0.2

LAKE
ONTARIO

0.2
LAKE
HURON

0.2

LAKE
MICHIGAN

0.6
0.9

LAKE ST. CLAIR

LAKE
ERIE

0.5

U.S.A.

U.S.A.

HUDSON BAY

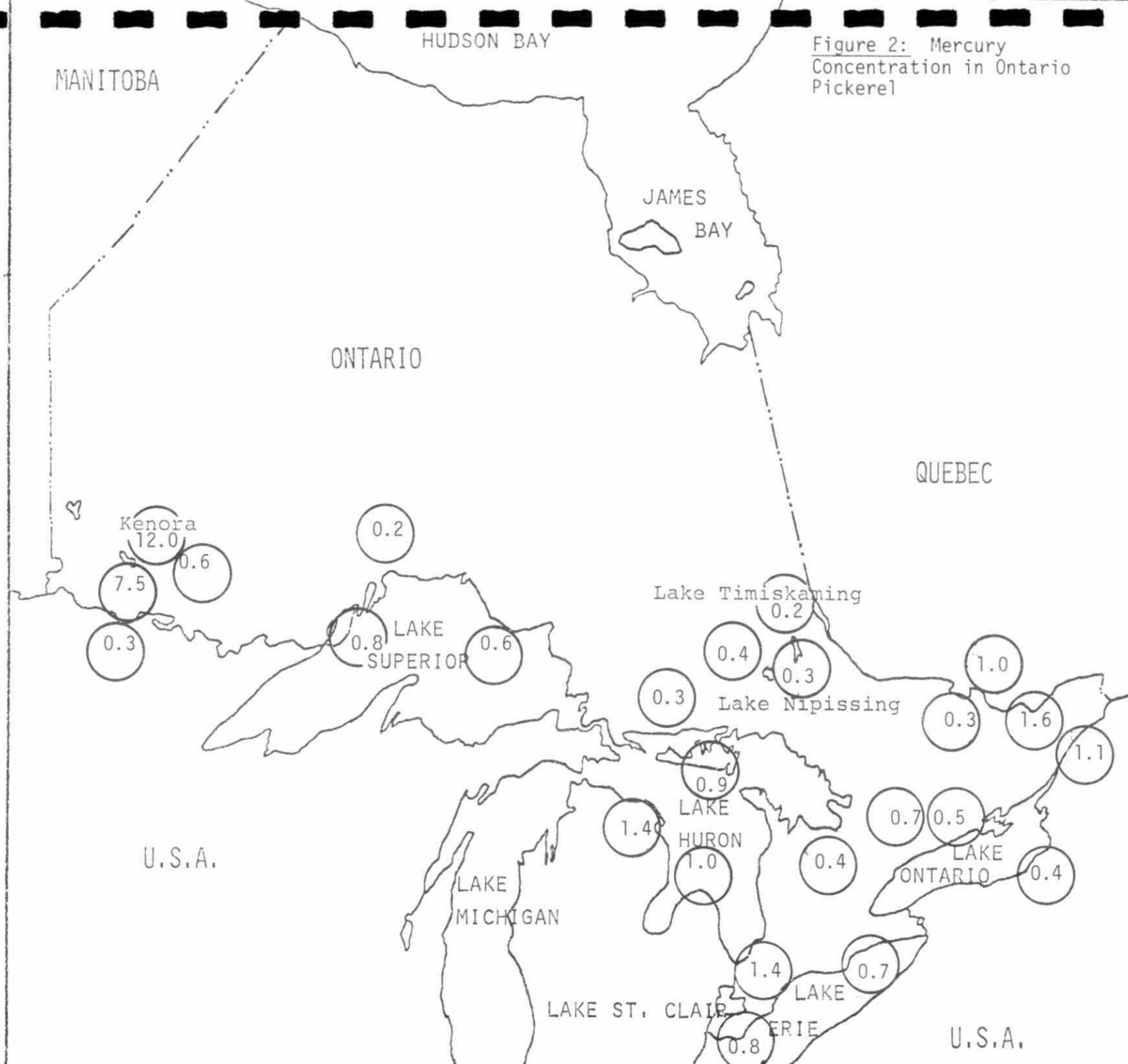
Figure 2: Mercury
Concentration in Ontario
Pickereel

MANITOBA

ONTARIO

JAMES
BAY

QUEBEC



HUDSON BAY

Figure 3: Mercury
Concentration in Ontario
Perch

MANITOBA

ONTARIO

JAMES
BAY

QUEBEC

Kenora

0.1

0.2

Lake Timiskaming

0.2

Lake Nipissing

0.2

0.7

0.6

0.4

0.2

0.3

0.2

0.4

LAKE
HURON

0.3

.7

0.1

LAKE
ERIE

0.3

0.6

0.2

2.8

0.3

LAKE ST. CLAIR

LAKE
MICHIGAN

U.S.A.

U.S.A.

TABLE 1: FRESH WATER FISH (MUSCLE TISSUE): ONTARIO (M.O.E. DATA)

AREA	RANGE (ppm)			AVERAGE (ppm)		
	N. PIKE	Y. PICKEREL	Y. PERCH	N. PIKE	Y. PICKEREL	Y. PERCH
ST. LAWRENCE RIVER: L. Ont. to Que. border	0.10-2.50	0.23-2.10	0.07-1.30	0.94	1.06	0.62
OTTAWA RIVER: Above Ottawa	0.15-0.82	0.08-0.60	0.10-0.23	0.23	0.26	0.16
OTTAWA RIVER: Below Ottawa	0.17-1.38	0.35-5.0	0.21-1.42	0.84	1.58	0.67
RIDEAU RIVER	0.12-1.3	0.44-2.5	-	0.41	1.04	-
BAY OF QUINTE AND EASTERN BASIN	-	-	0.24-0.79	0.65	-	0.44
MOIRA RIVER (SKOOTAMATTA)	0.28-0.68	0.24-0.71	0.13-0.16	0.48	0.53	0.15
MOIRA LAKE	0.24-1.0	0.47-1.51	0.34-0.35	0.60	0.68	0.35
LAKE ERIE	0.27-0.75	-	-	0.45	-	-
LONG POINT BAY	-	-	0.00-0.12	-	-	0.05
LAKE ERIE: WESTERN BASIN	-	0.36-1.33	0.09-0.77	-	0.83	0.32
LAKE ST. CLAIR: ST. CLAIR RIVER	0.12-4.3	0.19-5.3	1.6-5.3	0.87	1.41	2.80
LAKE SIMCOE	0.14-0.31	-	-	0.24	0.40	0.65
GEORGIAN BAY	-	-	0.13-0.42	-	-	0.24
LAKE HURON	0.21-0.27	0.30-3.00	0.02-0.68	0.24	0.89	0.28
THUNDER BAY (LAKE SUPERIOR)	0.76-1.39	0.63-0.94	-	1.05	0.78	-
LAKE NIPISSING	0.06-0.85	0.05-1.70	0.07-0.58	0.21	0.31	0.22
SUDBURY: FRENCH RIVER	0.16-0.51	0.09-0.64	-	0.32	0.26	-
COCHRANE: GERALDTON	0.04-0.86	0.03-0.66	0.00-0.15	0.27	0.21	0.09
FORT FRANCES: RAINY RIVER	0.22-0.65	0.19-0.35	-	0.42	0.27	0.28
KENORA	-	-	-	6.0	-	-
CLAY LAKE	-	7.0-8.0	-	-	7.5	-

TABLE 2:

FRESH WATER FISH (MUSCLE): ONTARIO

VARIOUS REFERENCES

AREA AND REFERENCE	RANGE (ppm)			AVERAGE (ppm)		
	N. PIKE	Y. PICKEREL	Y. PERCH	N. PIKE	Y. PICKEREL	Y. PERCH
LAKE ERIE: Chem. & Eng. News Ont. Lands & Forests Bureau of Comm. Fish Carr. R. L.	1.40-3.57	0.30-1.49	0.32-1.70 0.06-2.26 0.13-0.45		0.69	0.56 0.25
ST. CLAIR RIVER: Chem. & Eng. News			0.32-1.70	0.64		
LAKE ERIE: WESTERN Jervis, R. E.			0.21-0.26			
LAKE ST. CLAIR: Jervis, R. E.	0.32-0.63	0.58-0.66				
LAKE ONTARIO: Ont. Lands & Forests Jervis, R. E.	0.10-1.37	0.07-2.22	0.04-0.89	0.73 0.21	0.41	0.33
S. LAKE HURON: Bur. of Comm. Fish.	0.21-0.27	0.30-5.20 0.43-5.43	0.02-0.35	0.24	1.04 1.41	0.21
LAKE SUPERIOR: Bur. of Comm. Fish.	0.27-0.56	0.35-0.96	0.25-0.46	0.40	0.59	0.34
LAKE NIPISSING: Jervis, R. E.					0.42	0.20
ENGLISH RIVER: Ont. Lands & Forests	0.16-1.09	0.06-1.55		0.52	0.44	
WABIGOON RIVER: Ont. Lands & Forests	0.17-1.64	0.16-0.85		0.77	0.63	
CLAY LAKE: Scott, D. P.				8.87		
GRASSY LAKE: (N.W. ONT.) Scott, D. P.				3.30		
BALL LAKE: (N.W. ONT.) Scott, D. P.				3.66		
SEPARATION LAKE: (N.W. ONT.) Scott, D. P.				4.05		

FIGURE 4: Sampling Location



MERCURY LEVELS IN LAKE ST. CLAIR FISH

Species	1970-71			1973		
	No.	Range (ppm)	Mean (ppm)	No.	Range (ppm)	Mean (ppm)
Northern Pike	7	4.3-9.9	6.2	69	1.8-9.9	5.0
Perch	30	0.7-3.2	1.9	65	0.13-3.3	1.1
Rock Bass	8	2.6-8.1	5.7	63	0.22-4.25	1.69
Walleye	90	0.2-14	2.1	97	0.27-5.8	1.19

As was expected the mercury levels in Lake St. Clair area water samples were undetectable (0.001 ppm, which was the detection limit in 1970-71) except in regions where an industrial or municipal outfall was nearby.

ii - Declining Mercury Levels in Fish from the St. Clair System

Since 1970 various intensive surveys have been carried out involving the analysis of fish, sediments, water and aquatic life (plant and animal). One of these, the Baseline study, has been repeated on an annual basis, and has involved the analysis of mercury in approximately 20,000 fish. This data represents the mercury content of 30 different species which were categorized according to age, weight and length. This study has indicated that mercury levels are declining for many species.

For example, in 1970, a three year old walleye contained, on the average, 2.0 ppm mercury. In 1973, a three year old walleye contained (average) 0.97 ppm mercury.

For larger fish, e.g., six year old walleye, the level in 1970 was 4.0 ppm (average); whereas in 1973 it was 1.8 ppm (average). Thus the levels are dropping significantly if one compares fish of the same age over the past four years.

Comparing the same year class of fish as they become older (represented by successive annual catches in the Baseline Survey) shows another trend. A four year old fish in 1970 would have averaged 2.8 ppm mercury. In 1971, the same fish (now five years old) would have had about 3.0 ppm (average) mercury. In 1972, it would be six years old and would average 2.4 ppm mercury; in 1973, the same fish (seven years old) would average

2.3 ppm mercury. Thus the trend when one compares year class to year class, is that each class maintains their mercury concentration, or decreases in terms of mercury content only very slowly.

Accordingly, it seems that young fish today are starting out with less mercury per body weight and that this level remains roughly the same throughout the fish's life, or declines very slowly. Thereby, as the older, larger, and more heavily contaminated fish die out, the levels should continue to decline. It is hoped that the 1974 data will confirm these preliminary findings. Sampling for the 1974 Base-line Survey fish began in October 1974, arrived in the laboratory by mid-November, and analyses and interpretation of data should be complete by the end of March, 1975.

It is possible to establish a relationship between mercury concentration and fish length, for most species. Using the 1973 data, no fish (of any of the twelve species examined, including Pike, Large Mouth Bass, Perch, Small Mouth Bass, Walleye, etc.) over 20 cm, in length could be accepted as being safe for human consumption (that is, below 0.5 ppm) with a reasonable (95% confidence) safety factor.

iii - Breeding of Fish

The breeding operation appears to be strictly a commercial venture involving Purdy's and Olmstedt's fisheries. Fish caught in the St. Clair area and Southern Lake Huron are mixed with bread crumbs or similar substances in a ratio to bring the mercury concentration to less than 0.5 ug/g. As far as the Laboratory Branch Mercury staff can ascertain by telephone inquiries, no government agency is analyzing the final product and in the past Olmstedt's laboratory has had a bad record as far as mercury analysis is concerned.

The final product, breaded fish, almost certainly exceeds the mercury tolerance level set by many countries for their foodstuff, as can be seen in the following table:

<u>Country</u>	<u>Food</u>	<u>Tolerance</u> <u>(ppm Hg)</u>
Australia	All	0.1
Benelux Nations	All	0.03
Brazil	All	0.05
U.S.A.	Fish	0.5
U.S.A.	Fruits, vegetables	"zero"
Canada	Fish	0.5
Sweden	Fish	0.5

In any event, it remains to be clarified whether the

breeding operation is a means of creating a marketable form of mercury contaminated fish, or whether it merely creates adulterated bread.

III - MERCURY IN MUSEUM SPECIMENS: STUDIES

J. E. Paholeimo prepared a report for MOE regarding mercury levels in modern and museum fish. In summary, the laboratory produced about 2,100 analyses of fish captured after 1970, and about 500 analyses of museum fish that had been preserved for varying lengths of time.

This report takes a large body of analytical data on both present-day and preserved fish specimens, and using elementary statistical methods, demonstrated that the level of mercury in fish from Lake St. Clair and Lake Erie had significantly increased in recent years. Dr. Paholeimo used all of the museum fish data for which there were data from present-day specimens of the same species. By first demonstrating that there was no significant difference in the mercury concentrations of fish from Lake St. Clair and Lake Erie in historical times, he was able to compare historical specimens from Lake Erie with present-day specimens from Lake St. Clair. He then concluded that there were significant increases for almost every species in muscle mercury concentration.

Present day (1970-71) fish in Lake St. Clair show statistically higher levels of total mercury than do museum specimens from either Lake St. Clair or from Lake Erie. The 1960-1969 specimens from Lake St. Clair show higher mercury levels than the 1950-1959 Lake St. Clair specimens, and the recent (1970-1971) Lake St. Clair specimens are higher than the 1960-1969 specimens, with respect to total mercury content.

To date, there is one published report that reaches similar conclusions. This report, by Evans, Bails, and D'Itri (27) is based on mercury analyses of 57 fish, representing 26 species. The MOE Laboratory analyzed some of these fish, in support of the project. Evans et al placed all of the fish into four categories according to the mean level of mercury found in their muscle tissues, and concluded that "mercury levels in fish from Lake St. Clair and Lake Erie have been increasing since the early 1940's and the rate of increase accelerated in the late 1960's", and that "human health problems associated with mercury levels in fish are relatively recent".

A number of other researchers have analyzed preserved fish, both marine and freshwater, and have reached completely different conclusions.

Earl J. Harris and R. W. Karcher (28) analyzed seven

walleyes captured in 1939 in Lake Ontario, and nine walleyes captured in 1929 in Ticonderoga Creek, a tributary of Lake Champlain. These averaged 471 ppm mercury, respectively. Walleye from Lake Champlain analyzed in 1970 averaged 475 grams in weight and had a mercury concentration of 0.48 ppm. There were no walleyes captured in their 1970 survey of Lake Ontario, but comparisons are made to walleyes from the St. Lawrence River. These fish averaged 0.89 ppm, but were very large (average weight was 1855 grams). Harris and Karcher also examined several other areas in New York State, and conclude: "Although the buildup of mercury in the biomass, particularly in fish, has been thought of as a phenomenon of recent origin, the data presented in this paper would indicate this is an incorrect view" and that "those (fish) taken from Lake Ontario and Lake Champlain 30 or 40 years ago contained about as much mercury as they have in them today". They contend that natural levels and early industrialization contributed as much mercury to the environment near the beginning of the century as is contributed today.

Miller, Grant, Kishore, Steinkruger, Rowland, and Guinn (29) analyzed seven tuna that were caught between 1878 and 1909 and one swordfish that was caught in 1946, by neutron activation. They compared these data to analyses of recent tuna and swordfish. The mean mercury concentration (dry-weight basis) for the seven museum tuna samples was 0.95 ± 0.33 ppm. The corresponding mean for recent tuna was 0.91 ± 0.47 ppm. The museum swordfish result (1.36 ± 0.31 on dry-weight basis) falls within the range of values (0.94 to 5.1 ppm) found for modern swordfish. They conclude that "it is evident that there is no significant difference in mercury concentration between the museum tuna samples, caught 62 to 93 years ago, and the samples of tuna caught recently". Also, they looked for, but did not find, evidence of contamination of museum specimens by mercury, and contend "the data for both tuna and swordfish lend support to the contention that the mercury levels now being found in wide-ranging ocean fish are not primarily the consequence of man-made pollution, but are of natural origin". They point out the interesting estimate of Hammond (30) that if the TOTAL amount of mercury processed by man since 1900 were put into the world's oceans and mined, the average mercury concentration of seawater (about 0.1 ppb) would be increased by - at the most - 1%.

In another study based on marine species, Barber, Vijayakumar and Cross (31) reached conclusions similar to those of Miller et al (29). They examined benthopelagic fish -very deep feeding and deep living creatures.

Analysis of museum specimens of two species (one specimen each) for mercury (total mercury only) yielded data about the same as for mercury analysis of modern specimens of each species. Barber et al also perform a few regression line fits for their skimpy data, and on the basis of only twenty fish, state "concentrations exceeding 0.5 ppm (wet weight) may occur 'Naturally' in some species and may not be related to anthropogenic inputs of Hg to the oceans".

Methylmercury in fish was the topic of a paper by Zitko et al (32) who analysed an eel that had been preserved for 46 years and compared his finding to modern eel data. They concluded that the data indicated that "concentration of methylmercury has changed little, if at all, over a period of at least 46 years".

Wilmsen and Meyers (33) obtained prehistoric samples of channel catfish and freshwater drum, and analyzed them by neutron activation analysis. Their samples were of different ages, but were generally either dated at 100 B.C. or 400 A.D. They also analyzed tuna and anchovy from Chile, believed to have died around 1200 A.D., and they conclude: "Although these results are not definitive, they are mostly of comment ... the data do, however, suggest that mercury concentration in prehistoric fish was of the same order of magnitude as that in living individuals".

It may be worth mentioning the paper of Kevorkian et al (34) who determined mercury in a large number of human tissues, which were preserved from 1913 to 1970. Intercalibration analyses among four participating laboratories indicated an "intolerable wide variation of results on identical specimens - blatant discrepancies among all four laboratories", but this did little to dampen the optimism of their conclusion: "The most important (trend) regarding possibly dangerous environmental pollution ... demonstrates unequivocally an extraordinary decline in mercury content in almost all organs studies (sic) sharper in the early decades of this century, leveling off recently. Assuming that such tissue content is an expression of the relative degree of general environmental content or "pollution" ... the only tenable deduction is that our environmental sources are also diminishing".

The published information, then, is generally not in agreement with our own findings. It is noteworthy that most of the published information that does not agree with our position is based on an exceedingly small amount of raw data, or, as is the case with

Kevorkian et al (34), is based on contradictory, uncorroborated analytical data.

The papers dealing with marine species all indicate no change in mercury content in fish, with time. This may be due to the fact that in such an enormous water body, inputs that seem very large are, when balanced over the whole system, almost insignificant. Accordingly, only very large or very predacious fish are likely to become dangerously high in mercury content, and this has probably always been the case with such species. With freshwater species, in relatively confined water bodies, local influxes of mercury could have an altogether different effect. Lake St. Clair, for example, is known to have received considerable mercury due to industrial activity. In both the studies involving museum fish from this area, significant increases are demonstrated. For areas that have not received known large mercury inputs, no dramatic change in mercury levels in fish from different eras can be demonstrated.

For these reasons, I do not believe that the published data on other museum or prehistoric fish cast doubt on our findings, but tend to fill gaps in the total picture of the history of mercury contamination by man's industrial activity.

IV - METHYL MERCURY/TOTAL MERCURY RATIO

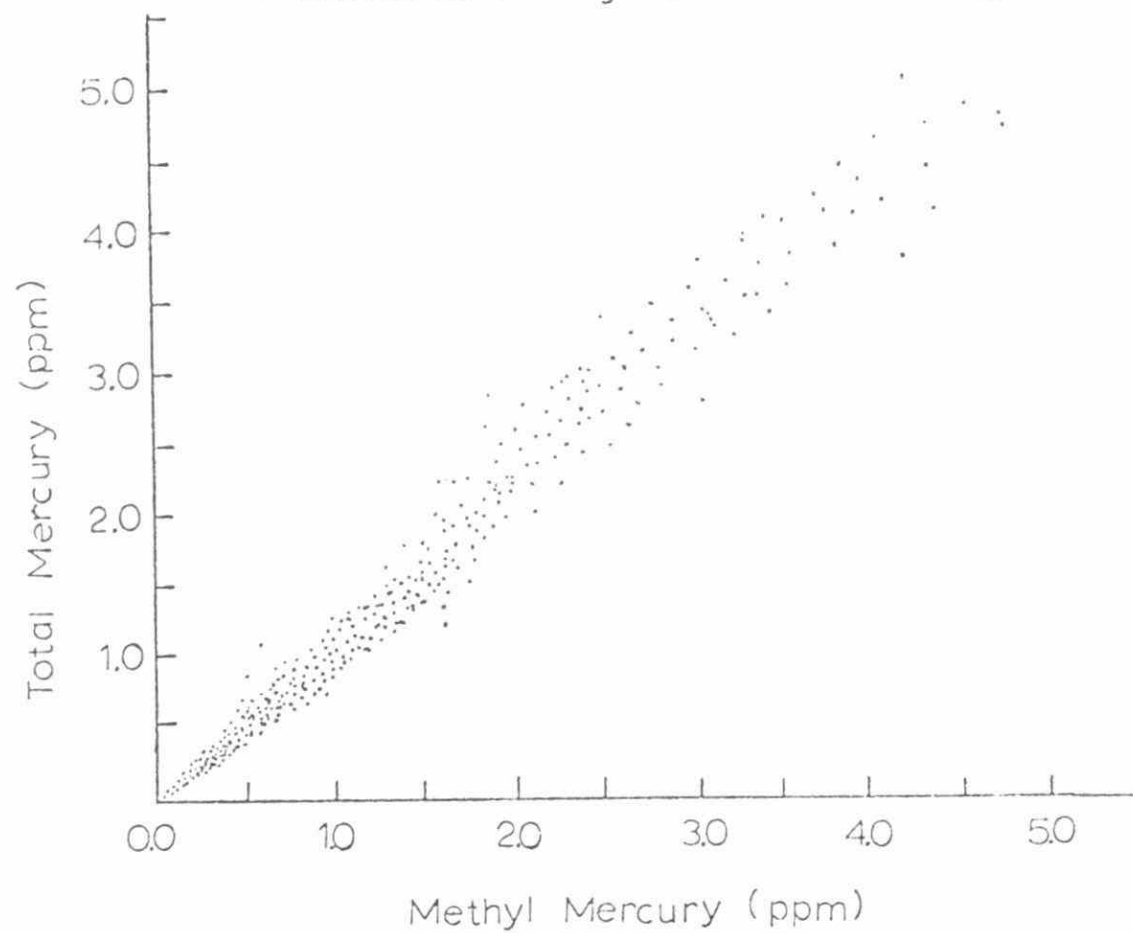
Nearly 600 fish representing 16 species were analyzed for methyl and total mercury. The average methyl to total mercury ratio was 88.9%, indicating that essentially all of the mercury in fish muscle is present as methylmercury. The effect of age and length on the methyl/total mercury ratio was examined, and it was found that these variables have no effect on the ratio. The ratio was similar for all sizes, ages, and species studied, and although some very small differences were noted between shallow, warm-water fish and bottom feeders, with respect to the ratio, the effect of industrialization on the ratio was found to be insignificant.

See Figures 5, 6, and 7.

FIGURE 5: METHYL/TOTAL MERCURY RATIO

TABLE I: MERCURY DATA				
Species	n	Total Hg Range (ppm)	Methyl Hg Range (ppm)	MeHg/Tot Hg Ratio %
Pumpkinseed	22	1.10-2.80	0.97-2.40	84.8 $\pm$ 9.8%
Bluegill	36	0.58-3.20	0.49-2.80	81.8 $\pm$ 8.7%
Perch	65	0.13-3.30	0.10-3.20	89.0 $\pm$ 11.0%
N. Pike	69	1.80-9.90	1.60-8.95	86.3 $\pm$ 11.0%
Bowfin	16	0.51-11.6	0.47-7.60	85.4 $\pm$ 14.0%
B. Crappie	36	0.79-3.70	0.64-3.30	85.4 $\pm$ 7.9%
LM Bass	14	1.04-4.12	1.07-3.87	90.2 $\pm$ 10.0%
N. Redhorse	28	0.07-5.16	0.07-5.00	92.1 $\pm$ 8.0%
Y. Bullhead	27	0.62-4.88	0.52-4.00	86.9 $\pm$ 7.9%
Channel Cat	11	1.05-3.94	0.99-3.55	92.0 $\pm$ 7.6%
Y. Walleye	97	0.27-5.80	0.29-4.50	88.5 $\pm$ 12.0%
F.W. Drum	22	0.11-1.89	0.11-1.68	95.1 $\pm$ 7.1%
White Bass	27	0.44-3.35	0.38-3.58	93.2 $\pm$ 10.0%
Muskie	13	1.08-23.3	0.92-20.4	88.0 $\pm$ 6.6%
Rock Bass	63	0.22-4.25	0.16-4.21	92.1 $\pm$ 8.2%
Carp	30	0.31-2.90	0.25-2.70	95.3 $\pm$ 10.0%

FIGURE 6: Methyl & Total Mercury



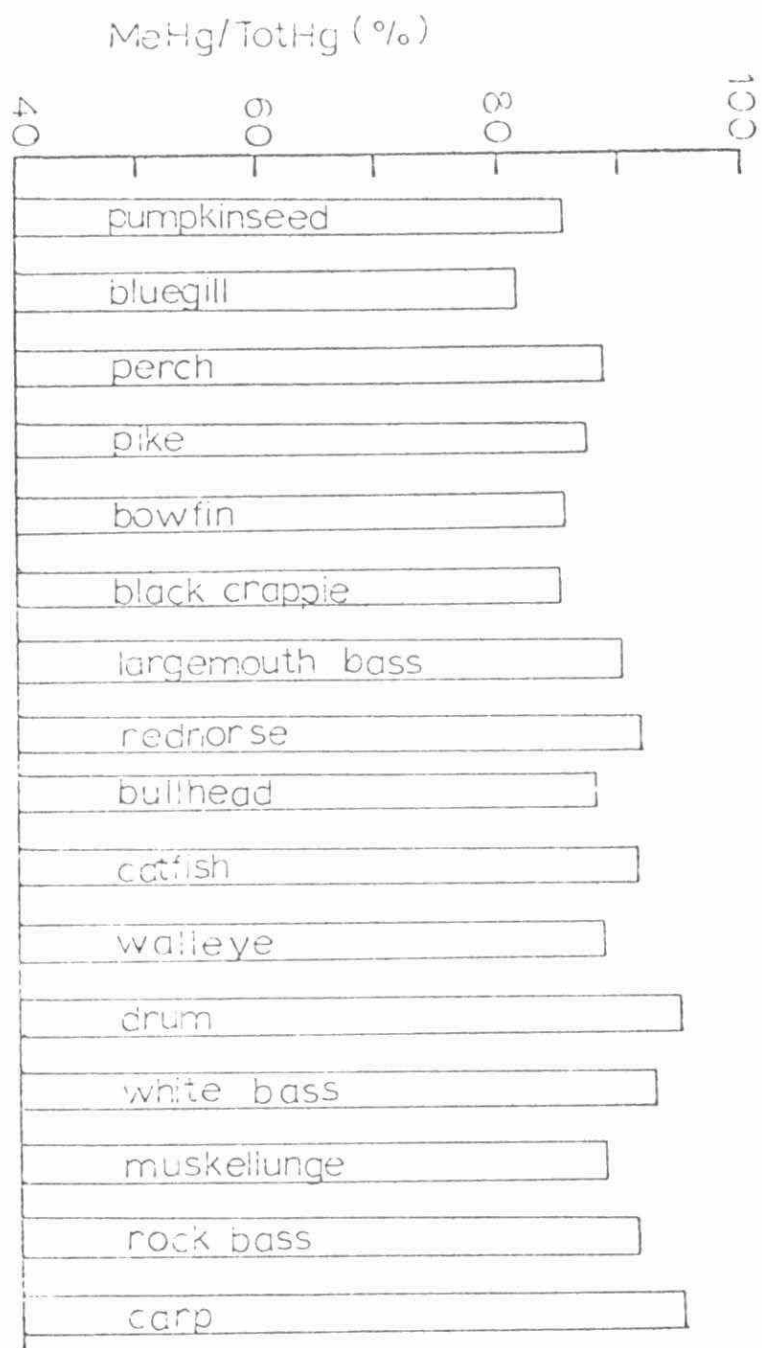


FIGURE 7: MeHg/TotHg by Species

V - SUMMARY OF MERCURY CONCENTRATIONS IN SEDIMENTS
AND FISH FROM THE ENGLISH RIVER - WABIGOON SYSTEM
1970-1974

Introduction:

The first known industrial input of mercury into the Wabigoon - English River system occurred in the early 1960's when the Dryden Chemical Co. was formed and opened a chlor-alkali plant (using mercury electrodes) to supply chemicals for the Dryden Paper Company's kraft paper mill situated in Dryden, Ontario. From the time the chlor-alkali plant began operation in 1962 to the time severe restrictions on the mercury content of industrial effluents were imposed by the Ontario government in 1970, the total loss of mercury from the plant has been estimated between 20,000 and 50,000 pounds.

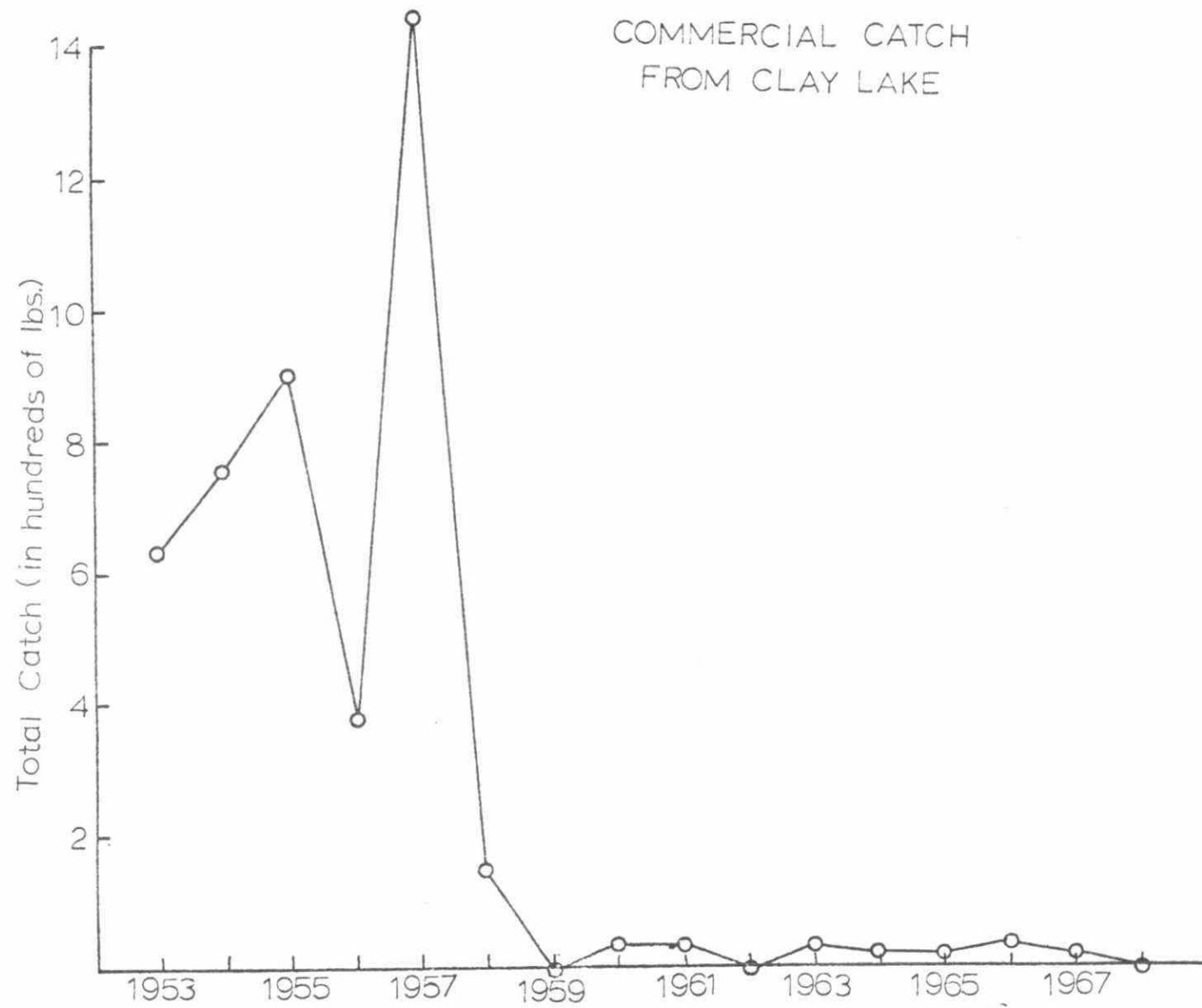
Fishermen around Clay Lake, some 60 miles downstream from Dryden, established a commercial fishery in the early 1920's. In 1957, however, effluents from the paper mill caused severe taste and odour problems, and the commercial fishing has been restricted to test catches ever since. (Fig. 8) The English River system however, was not seriously affected by paper mill wastes, and was extensively used for sport fishing.

Several lodges are situated along the English and Winnipeg Rivers, and largely employ Indians from the Whitedog and Grassy Narrows Indian Reservations as guides. These guides, and the Indian population in general, consume fish as a significant portion of their diet. The owners of the lodges are generally dependent on tourists whose primary interest is sport fishing.

General Description:

The area of concern is in north western Ontario to the north of Kenora. (Fig. 1) Dryden is situated on the Wabigoon River where it flows out of Wabigoon Lake. The Wabigoon River flows through Clay and Segise Lakes to join the English River at Ball Lake. The English River is actually a series of small lakes, and flows to Umfreville Lake, where it joins the Winnipeg River at Boundary Falls. The Winnipeg River flows from the Lake of the Woods to Lake Winnipeg in Manitoba. A detail of the Wabigoon River, with flow direction, from Dryden to Clay Lake is shown in Figure

Figure 8 :
COMMERCIAL CATCH
FROM CLAY LAKE



Mercury Concentrations:

a) Sediment

The effect of the Dryden Chemical Company on the mercury concentration in the sediment is clearly seen in Figs. 9 and 10. The mercury concentrations shown (in ug/g) are the average of three sediment samples for the 1970 data, and nine samples for the 1972 data. Sediment samples for the first twenty miles downstream of Dryden typically consist of wood fibres, due to input from the paper mills. The mercury concentration in these samples is typically quite heterogeneous, and a range in concentration in excess of a factor of ten is not unusual at most of the sites.

The sediment mercury levels in the portion of the Wabigoon River and from Clay to Launt Lakes are higher than the critical level of 0.3 ppm, claimed by Jernelov to be the lowest level in sediment which can demonstrably cause fish to reach levels in excess of 0.5 ppm in their muscle tissue.

b) Fish

Virtually no fish are present in the Wabigoon River downstream from Dryden to Clay Lake. Wabigoon Lake and tributary lakes and rivers support normal populations of fish.

The concentration of mercury in fish from Clay Lake are higher than for any other fish analyzed in North America. Some large yellow walleye have been measured to have concentrations in excess of 25 ug/g - more than 50 times the concentration deemed safe for human consumption. As can be seen in Figure 11, the concentration of mercury in walleye decreases somewhat further along the English and Winnipeg Rivers, but fish in excess of 4 ug/g are caught at the border of Manitoba. The effects of the mercury input at Dryden can be traced as far as Lake Winnipeg. The data for northern pike indicates the same trends as those for walleye, with the flow patterns in the river system being clearly reflected in the concentration of mercury in the fish. The concentrations indicated are the averages of at least three fish of each species, such as cisco and white sucker show the same trends, although the levels are somewhat lower (about 4-7 ug/g in Clay Lake).

c) Wildlife

Aquatic birds in the area also show elevated levels of mercury, with concentrations of mercury in excess of 15 ug/g having been measured in the flesh of ducks from Clay Lake. Mink, moose, and deer on Clay Lake have also shown concentrations in muscle in excess of 1 ug/g, probably due to the ingestion of contaminated fish or factors related to general mercury contamination in

Figure 9
 MERCURY IN WABIGOON-ENGLISH RIVER SEDIMENTS ($\mu\text{g/g}$)
 1971

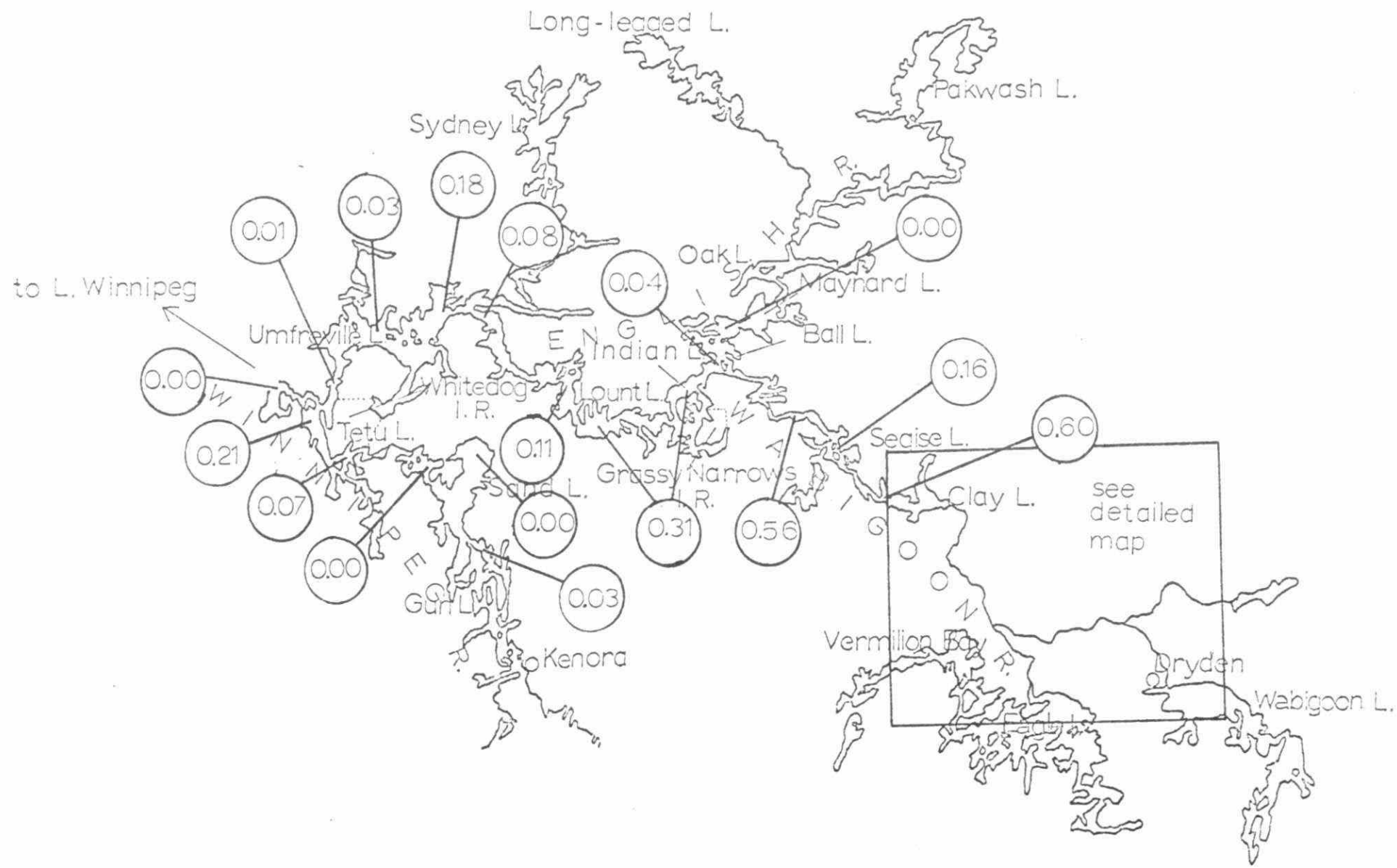


Figure 10

MERCURY IN WABIGOON SEDIMENTS ($\mu\text{g/g}$)

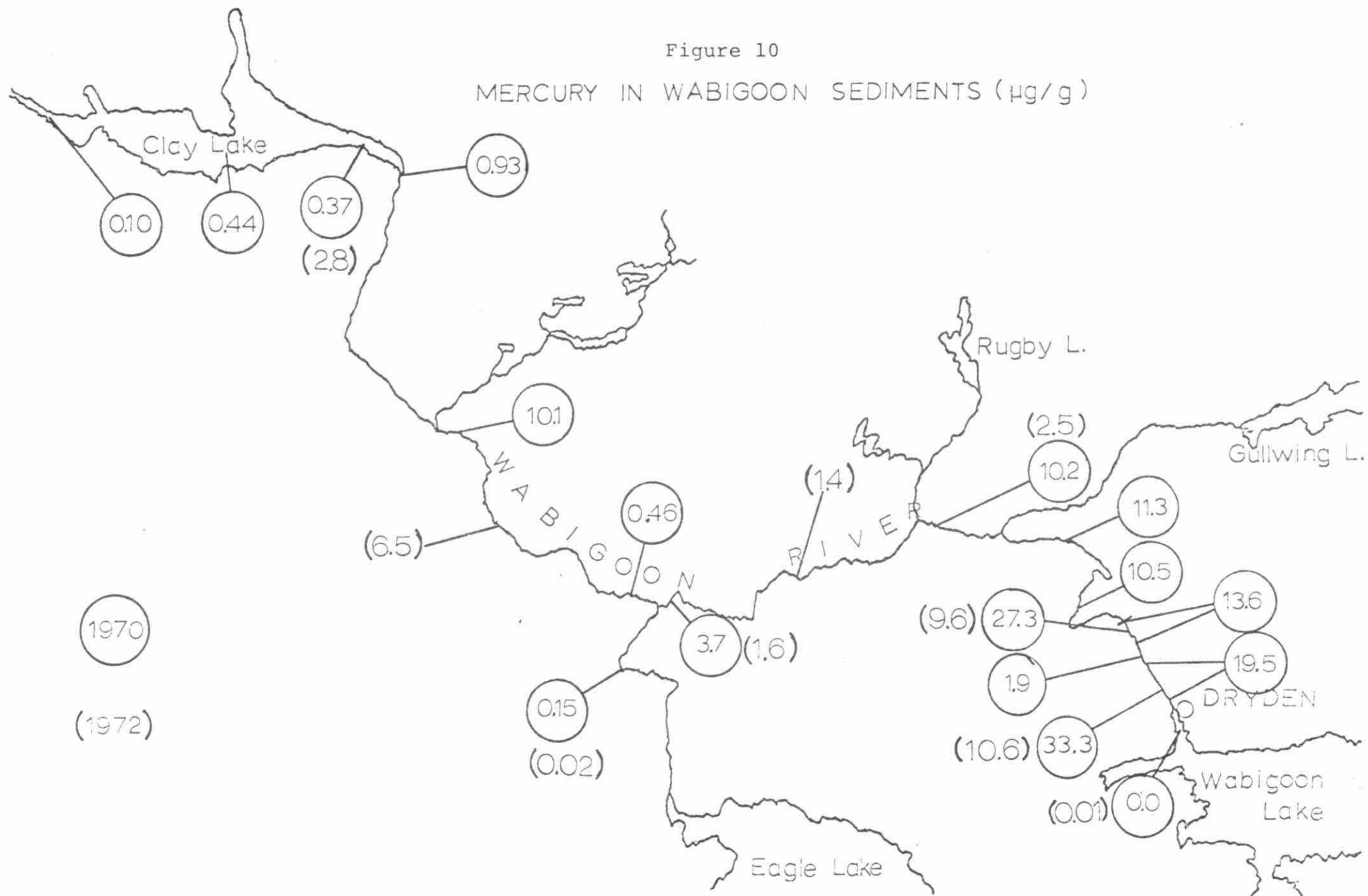
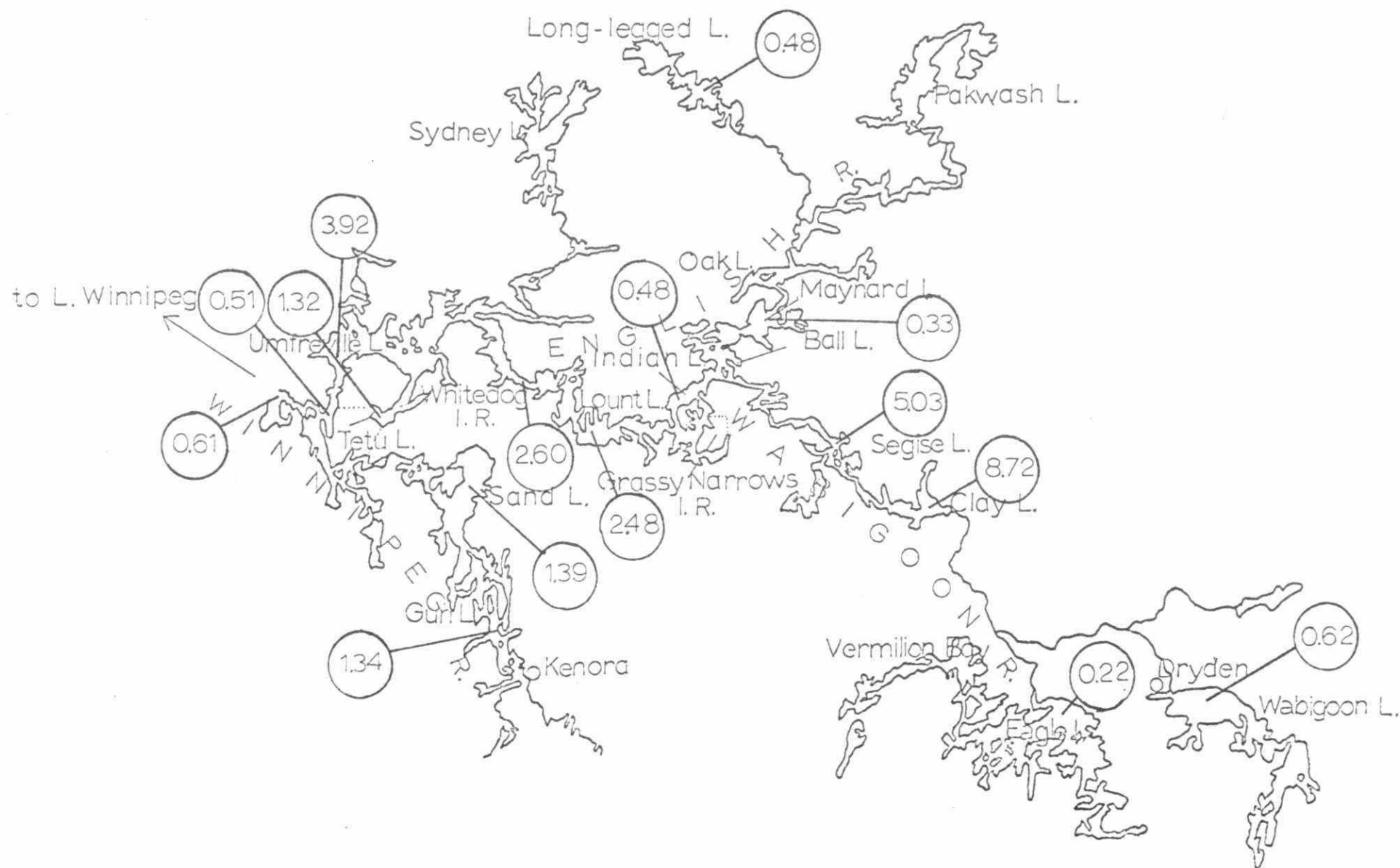


Figure 11

MERCURY IN YELLOW WALLEYE ($\mu\text{g/g}$)

1970



the area.

d) Humans

Indications of elevated levels of mercury in humans in the area appeared in 1970, when mercury concentration in the hair of 32 residents from the Kenora area showed levels that averaged 7-10 times the Canadian average. Some individuals had levels up to 30 times the Canadian average. Since then, several programs of analyzing the concentration of mercury in the blood of Indians and residents of the area have been carried out by the Ontario Ministry of Health and federal agencies.

D: SAMPLING AND ANALYSIS OF MERCURY

Sampling

WATERS: If clear, add enough Aristar or Ultrex HNO_3 to maintain pH of at least 1. Add enough 6% KMnO_4 to maintain pink colour.

If much particulate matter is present, add enough high purity HNO_3 to bring pH to 1, add enough KMnO_4 to maintain pink colour, plus 1 ml per 100 ml sample.

SEDIMENTS: Note location and sample description carefully. Try triplicate triangle sampling if unsure of homogeneity of river bottom. Ask for duplicate or multiple analyses in order to obtain precision data (standard deviations).

Most dredges are useless for reliable mercury work; the use of divers is recommended. Keep samples in glass jar (or plastic) - no metal should ever come into contact with the sample.

FISH, INSECTS, PLANTS, ETC.: For biological samples, keep fresh, freeze immediately, write thorough details of species, size, sex, location of capture, and clarify those organs or parts that you wish analyzed, and whether any analyses other than mercury are required. Package sample in clear bags and keep contents frozen. Tape or tie instructions on OUTSIDE.

ANALYSIS:

The MOE has prepared a booklet entitled "The Determination of Mercury in Environmental Samples". (35) This publication provides details of the procedures for analysis of mercury in water, industrial waste, fish, and sediment by flameless Atomic Absorption Spectrometry, as well as methylmercury in fish by Gas Chromatography. Persons interested in obtaining carbon copies of this publication should contact the author at: Inorganic Trace Contaminants Section, Laboratory Services Branch, P.O. Box 213, (Resources Road), Rexdale, Ontario M9W 5L1.

It is hoped that this presentation has clarified some of the aspects of the mercury situation, especially regarding analysis and actual levels throughout the Province. The need for more thorough monitoring and control of this potentially lethal element and its compounds is obvious, and this region - Northwestern Ontario - is one of the areas where our strictest attention must, and will be, focussed.

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